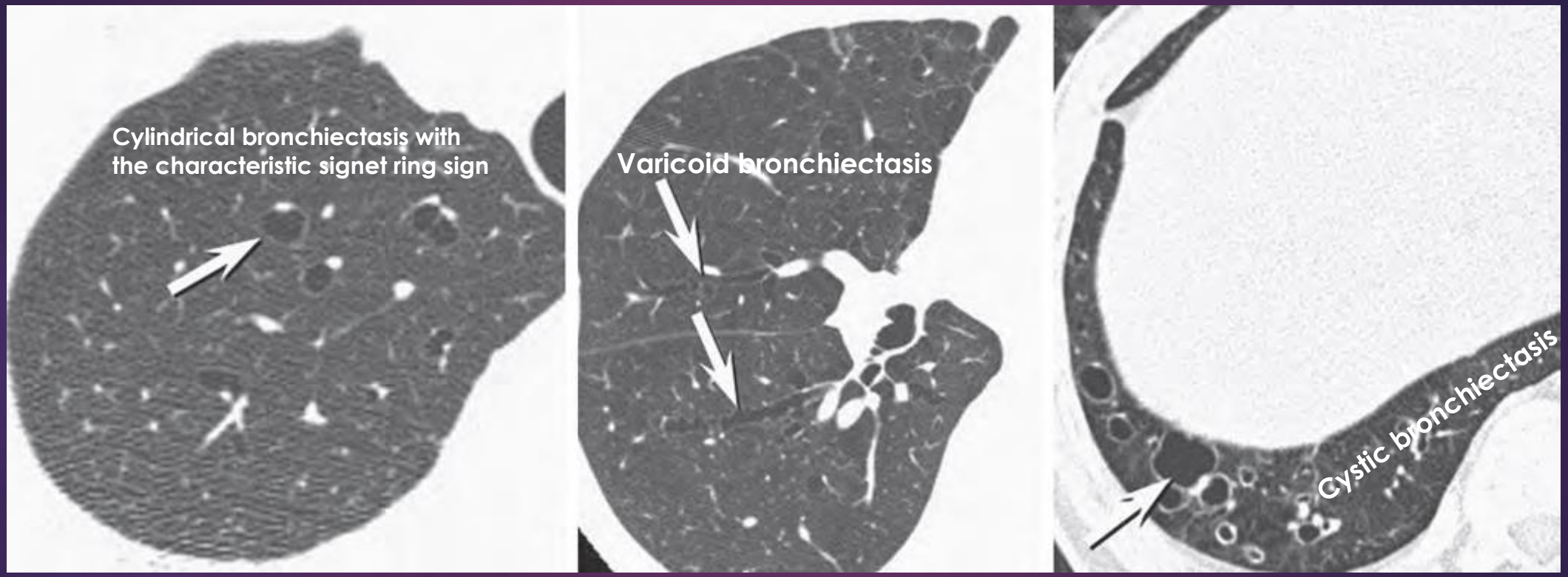
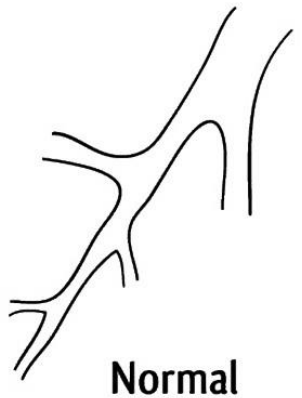
284 Bronchiectasis

Rebecca M. Baron, Miriam Baron Barshak

DR. M.FARZMEHDI

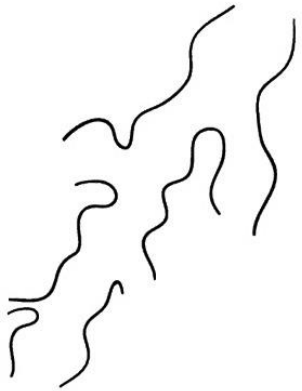
Bronchiectasis



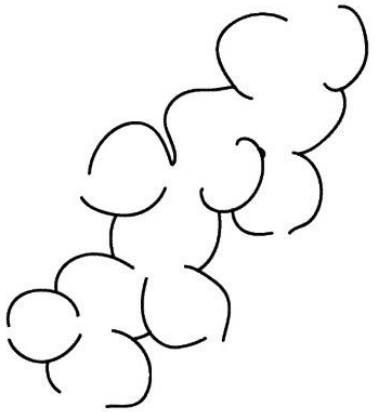


Normal

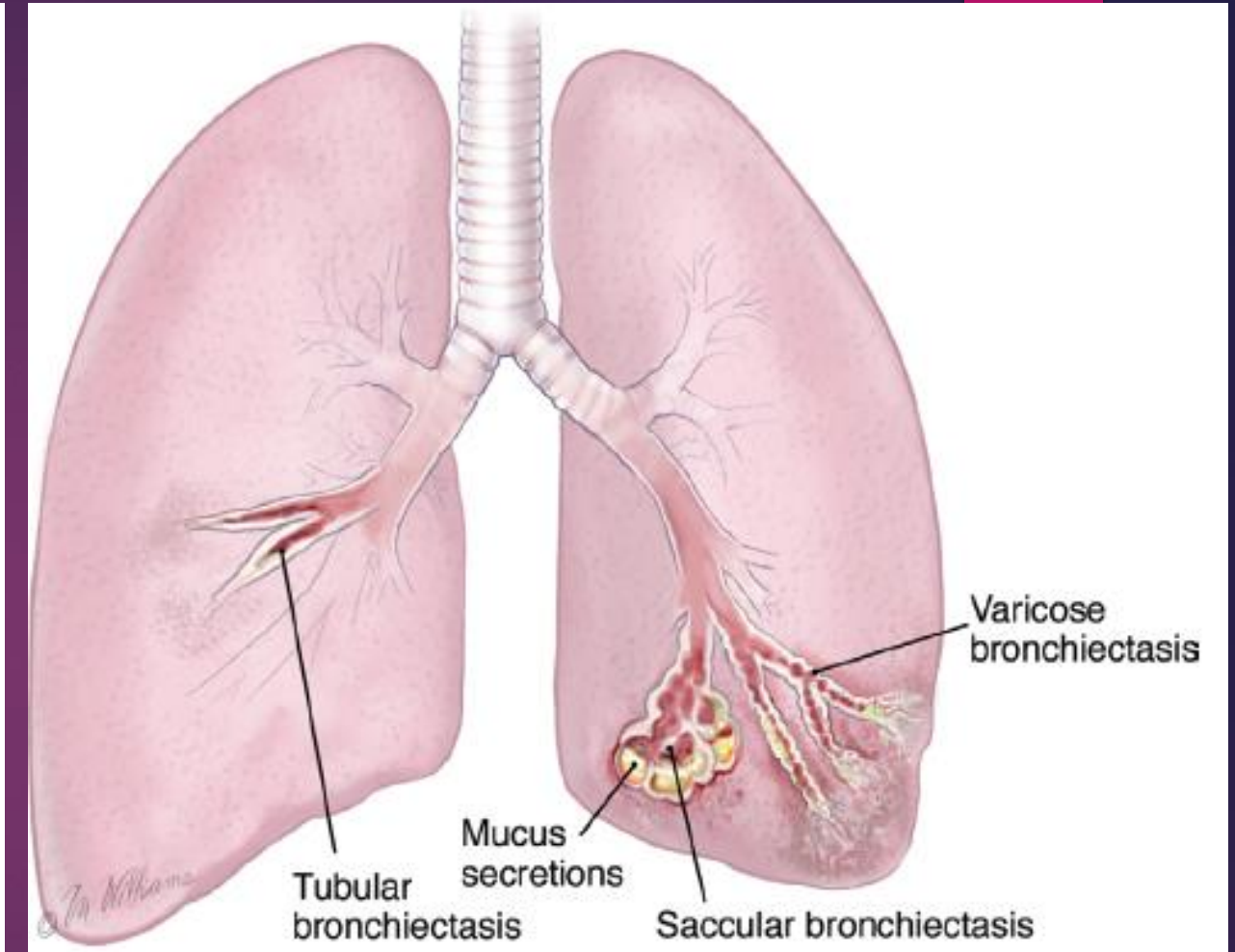
Cylindrical



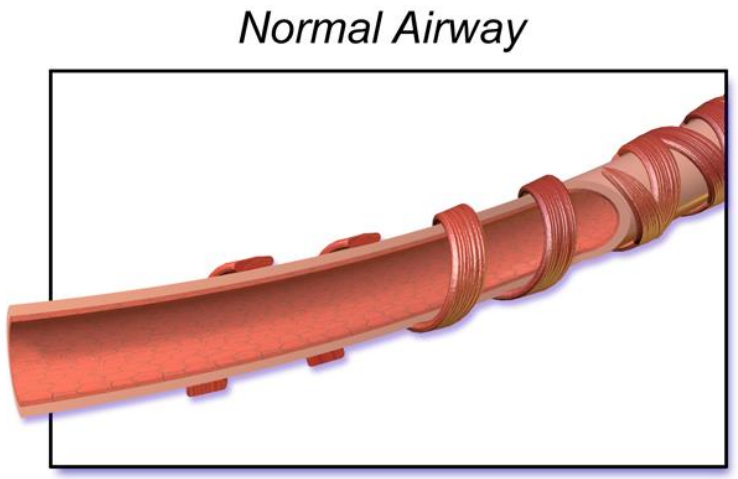
Varicose

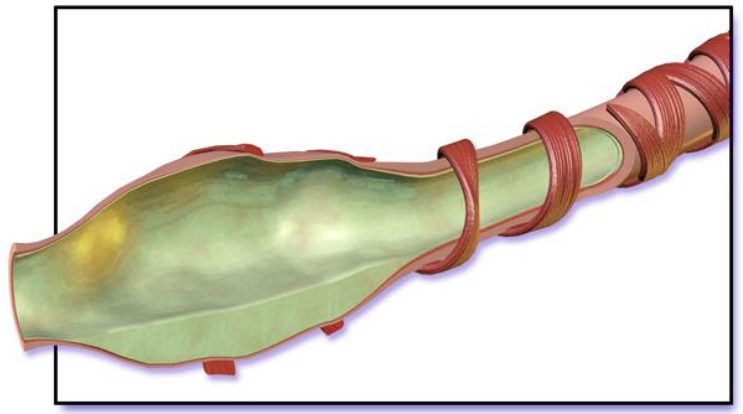


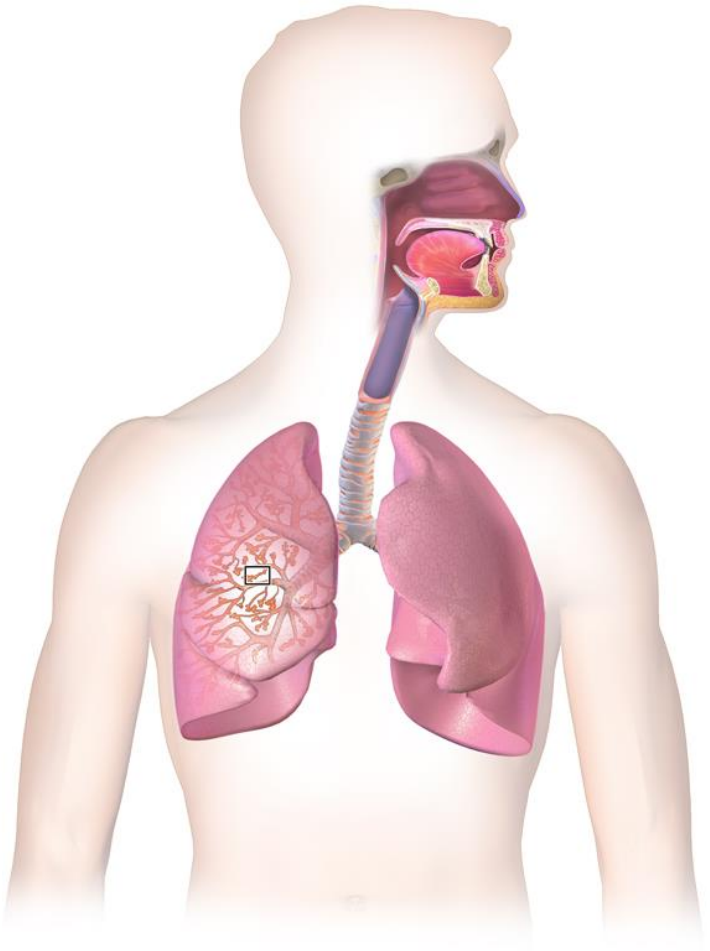
Cystic



Source: D. J. Sugarbaker, R. Bueno, Y. L. Colson, M. T. Jaklitsch, M. J. Krasna, S. J. Mentzer, M. Williams, A. Adams: *Adult Chest Surgery*, 2nd Edition: www.accesssurgery.com



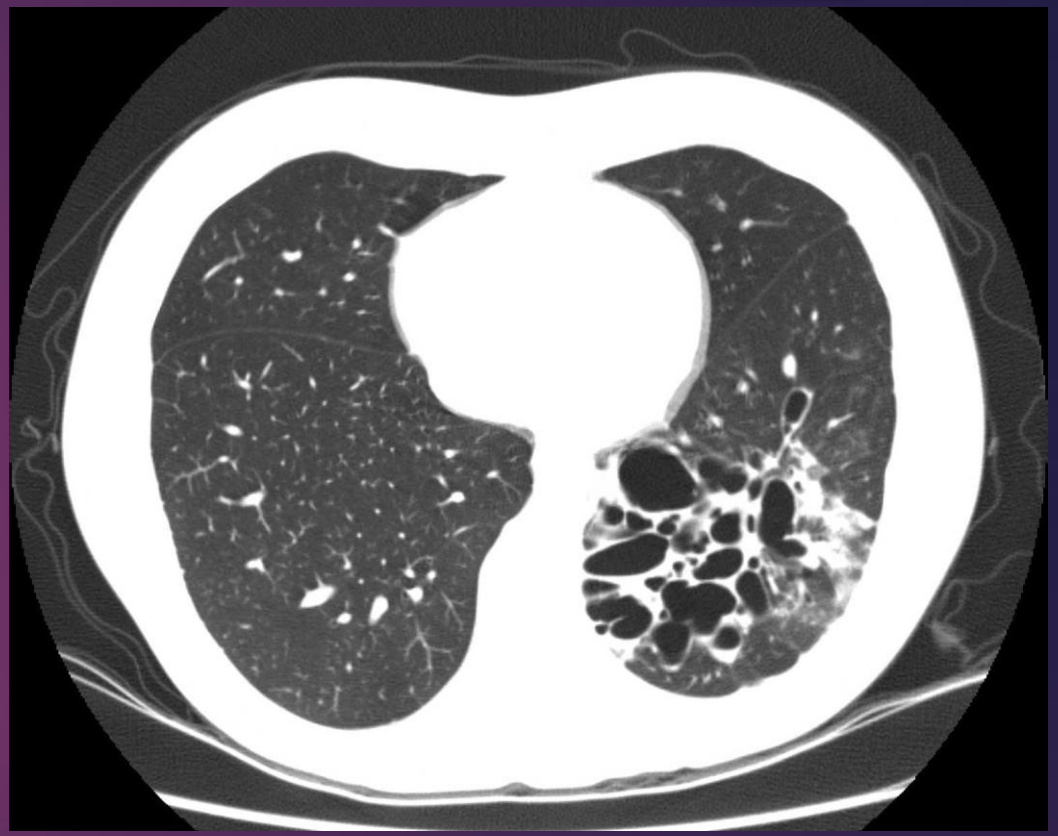




Bronchiectasis

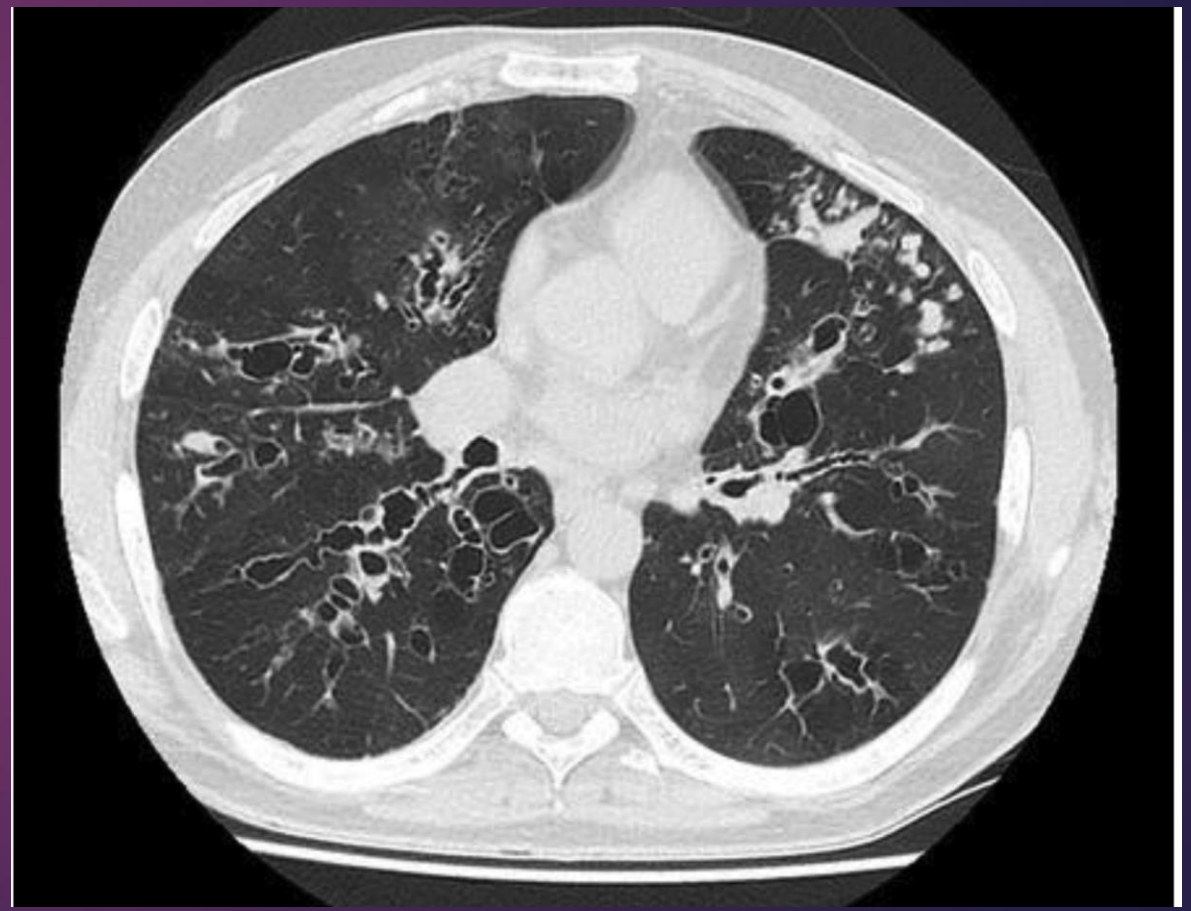
ETIOLOGY
Focal

- Obstruction
  - Extrinsic
    - lymphadenopathy
    - parenchymal tumor mass
  - Intrinsic
    - airway tumor
    - aspirated foreign body
    - scarred/stenotic airway
    - bronchial atresia
- Chest imaging (chest x-ray and/or chest CT);
- Bronchoscopy



ETIOLOGY
Diffuse

- Infectious
  - bacterial, TB , fungi
  - nontuberculous mycobacterial
  - Recurrent aspiration
- Noninfectious
  - Immunodeficiency
  - Genetic
  - Autoimmune or rheumatologic
  - Miscellaneous
    - Yellow nail syndrome
    - Traction bronchiectasis
      - postradiation fibrosis
      - idiopathic pulmonary fibrosis
  - Idiopathic 25–50%



ETIOLOGY
Diffuse: upper lung fields

- cystic fibrosis (CF)
- Postradiation fibrosis

ETIOLOGY
Diffuse: Lower lung fields

- ***Chronic recurrent aspiration***
  - esophageal motility disorders like those in scleroderma
- ***End stage fibrotic lung disease***
  - Traction bronchiectasis from IPF
- ***Recurrent immunodeficiency-associated infections***
  - Hypogammaglobulinemia

ETIOLOGY
Diffuse: midlung fields

- ***NTM***
  - Mycobacterium avium-intracellulare complex (MAC)
- ***Dyskinetic / immotile cilia syndrome***
- ***ABPA***
  - immune-mediated reaction to Aspergillus damages the bronchial wall
- ***Congenital***
  - cartilage deficiency
    - Tracheobronchomegaly (Mounier-Kuhn syndrome)
    - Williams-Campbell syndrome

EPIDEMIOLOGY Bronchiectasis

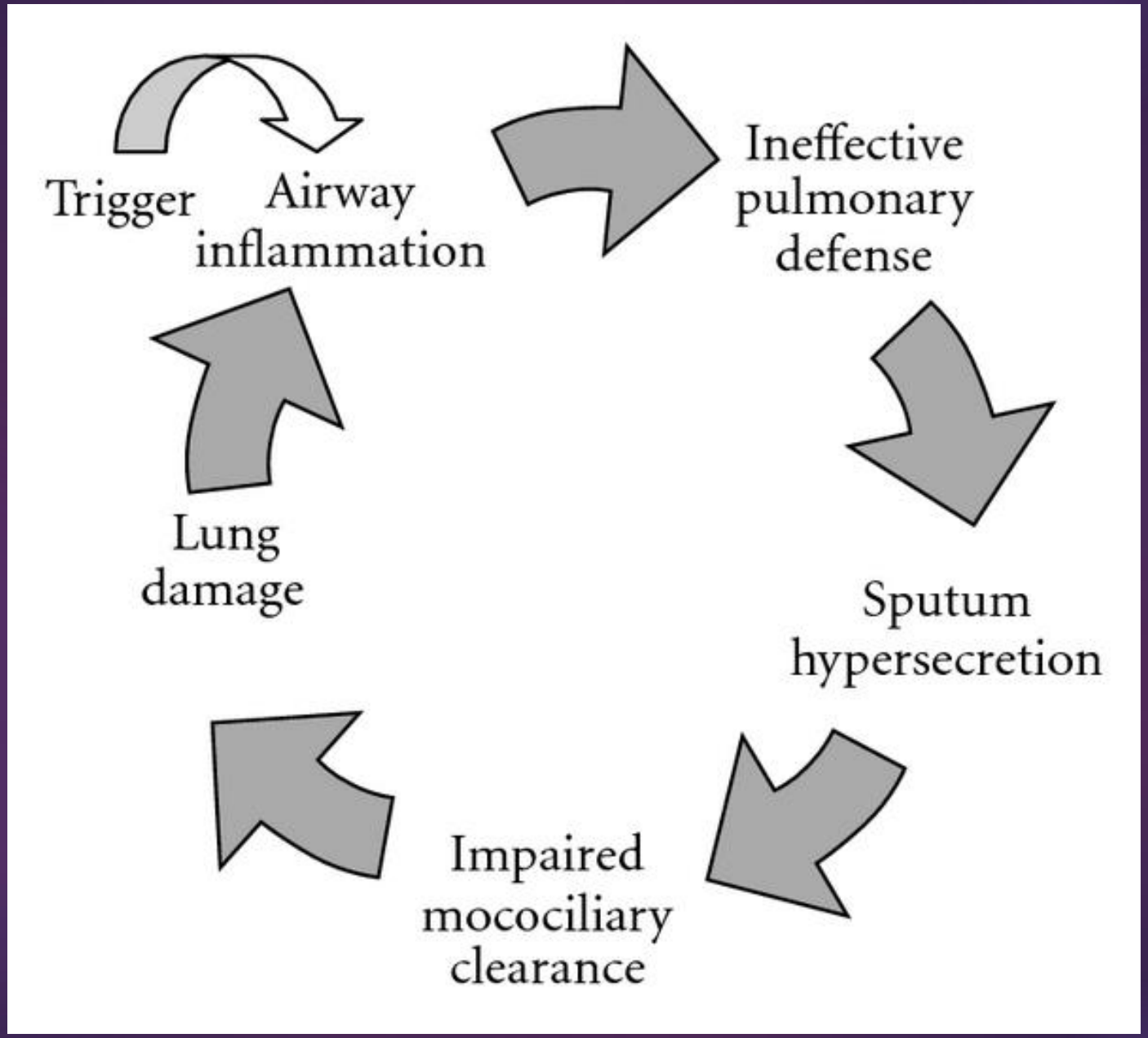
- Prevalence 
- Varies greatly with the underlying etiology
  - CF in late adolescence or early adulthood
  - Atypical CF in adults in their thirties and forties
- MAC infection
  - nonsmoking women >50 years of age
- incidence of bronchiectasis increases with age
- women > men
- reactivated tuberculosis
  - sequela of granulomatous infection
- increased incidence of non-CF bronchiectasis?

PATHOGENESIS AND PATHOLOGY
Bronchiectasis

- mechanism of infectious bronchiectasis
- ***"vicious cycle hypothesis,"***
  - susceptibility to infection
  - poor mucociliary clearance

result in

- microbial colonization of the bronchial tree


Trigger
Airway inflammation
Ineffective pulmonary defense
Sputum hypersecretion
Impaired mocociliary clearance
Lung damage

Pseudomonas aeruginosa

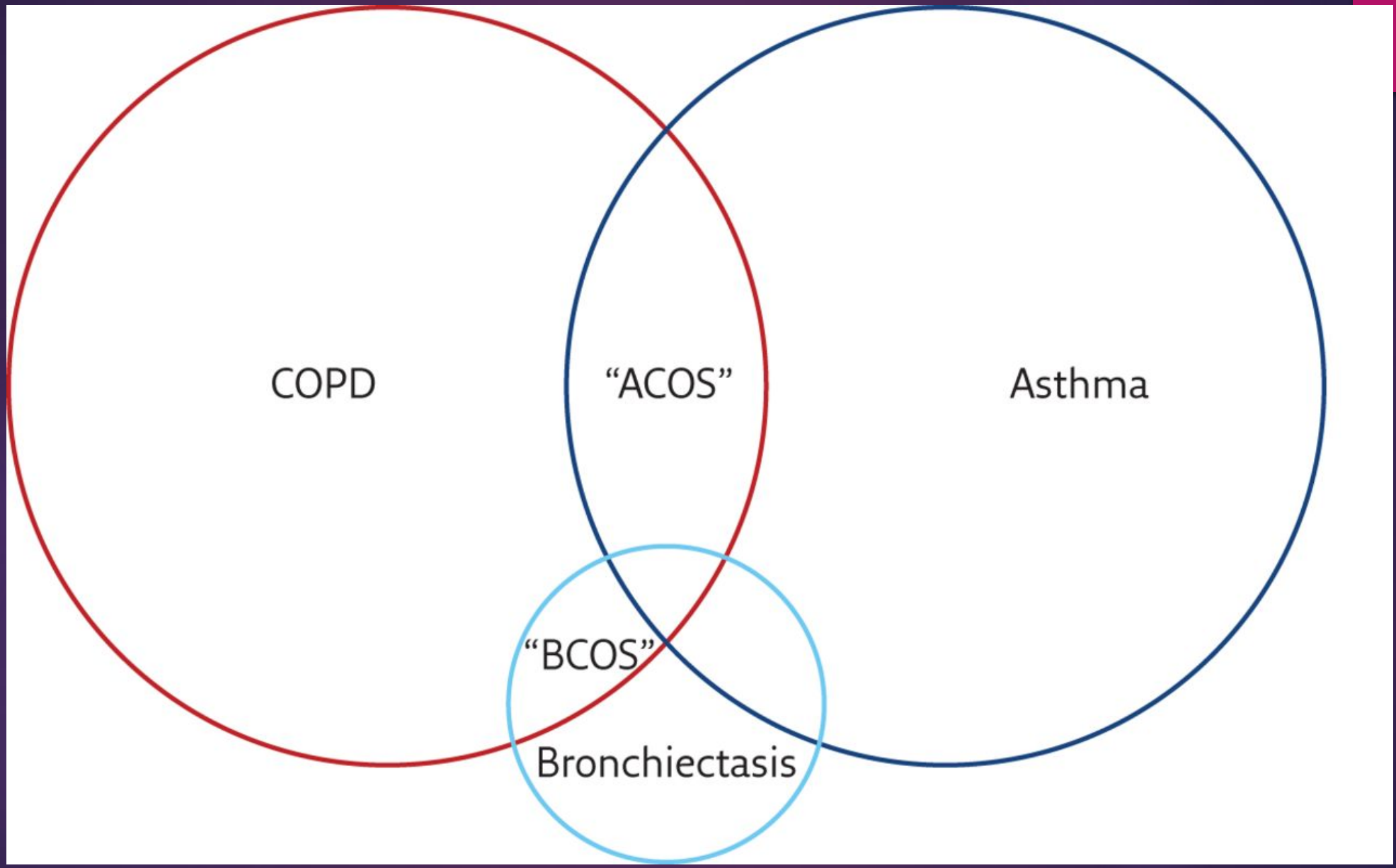
- colonizing damaged airways
- Evading host defense mechanisms
- Impaired mucociliary clearance

pathology of bronchiectasis

- significant small-airway wall inflammation
- larger-airway wall destruction as well as dilation
  - loss of elastin, smooth muscle, and cartilage
  - release proteases and other mediators
  - reactive oxygen species and proinflammatory cytokines
  - damage the larger-airway walls
- airflow obstruction
- Antiproteases
- a1 antitrypsin
  - neutralizing the damaging effects of neutrophil elastase
  - enhancing bacterial killing
- a1 antitrypsin deficiency → Bronchiectasis and emphysema

noninfectious bronchiectasis

- ***immune-mediated reactions***
  - Sjögren's syndrome
  - Rheumatoid arthritis
- ***Traction bronchiectasis a result of lung fibrosis***
  - postradiation fibrosis
  - idiopathic pulmonary fibrosis


COPD
“ACOS”
Asthma
“BCOS”
Bronchiectasis

CLINICAL MANIFESTATIONS

- Productive cough
  - thick, tenacious sputum
- crackles and wheezing on lung auscultation
- clubbing
- Mild to moderate airflow obstruction

CLINICAL MANIFESTATIONS
Acute exacerbations of bronchiectasis

- changes in the nature of sputum production
  - increased volume and purulence

- Fever and new infiltrates, may not be present

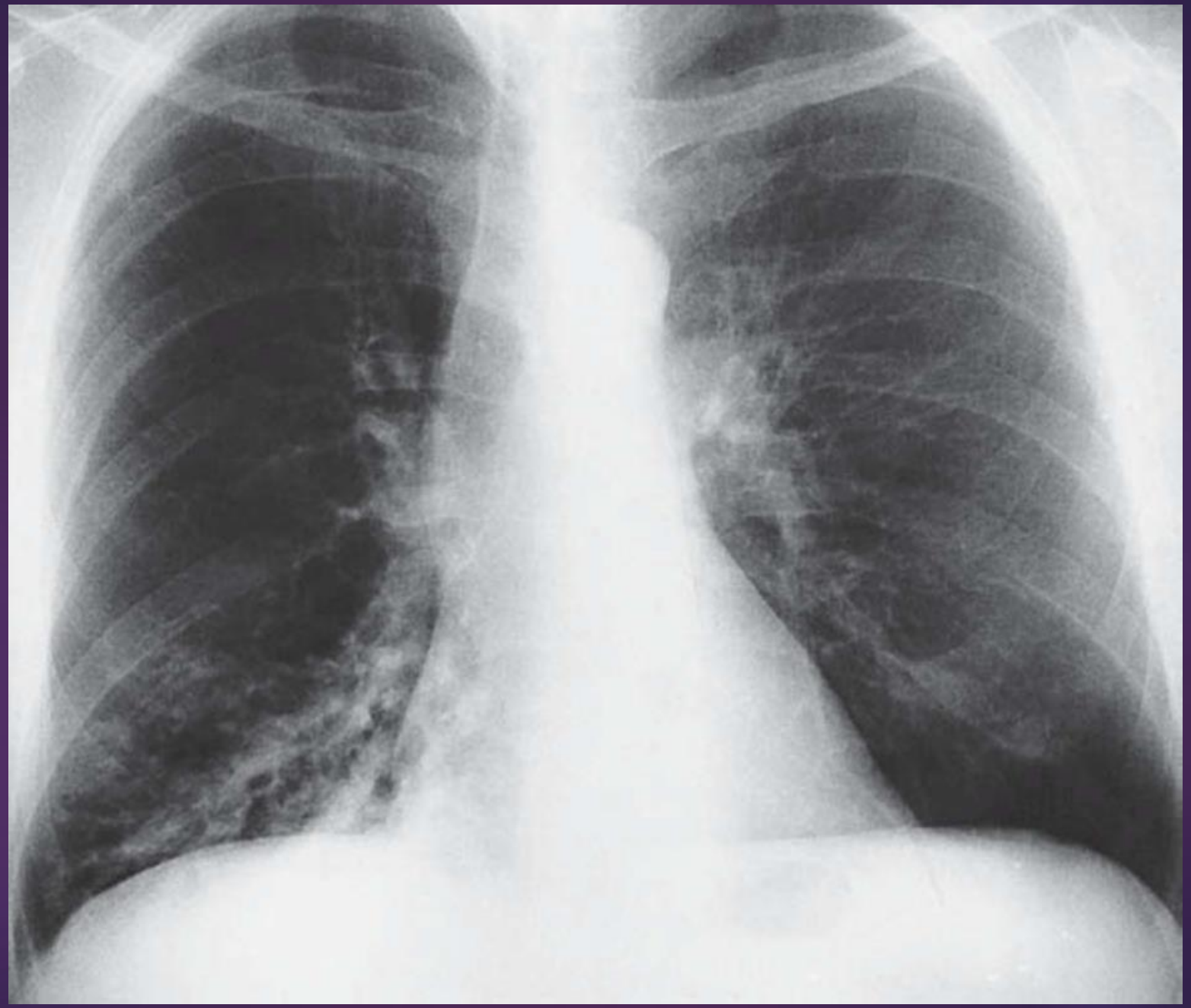
DIAGNOSIS

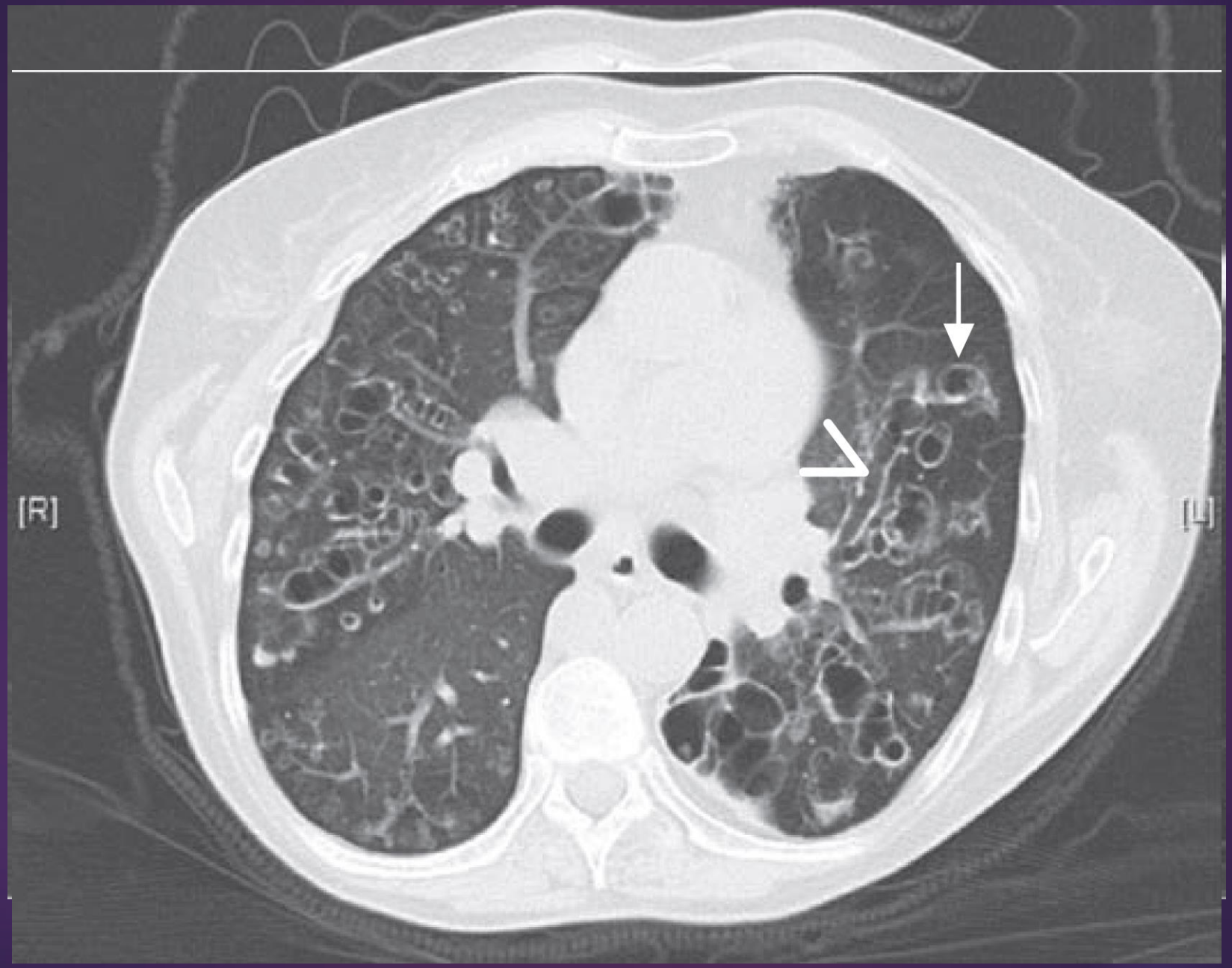
- Persistent chronic cough and sputum production

- Consistent radiographic features

DIAGNOSIS

- Chest radiographs lack sensitivity → "tram tracks"
- Chest CT
  - airway dilation
  - "signet-ring sign"
  - lack of bronchial tapering
  - bronchial wall thickening
  - Inspissated secretions ("tree-in-bud" pattern)
  - cystic bronchiectasis

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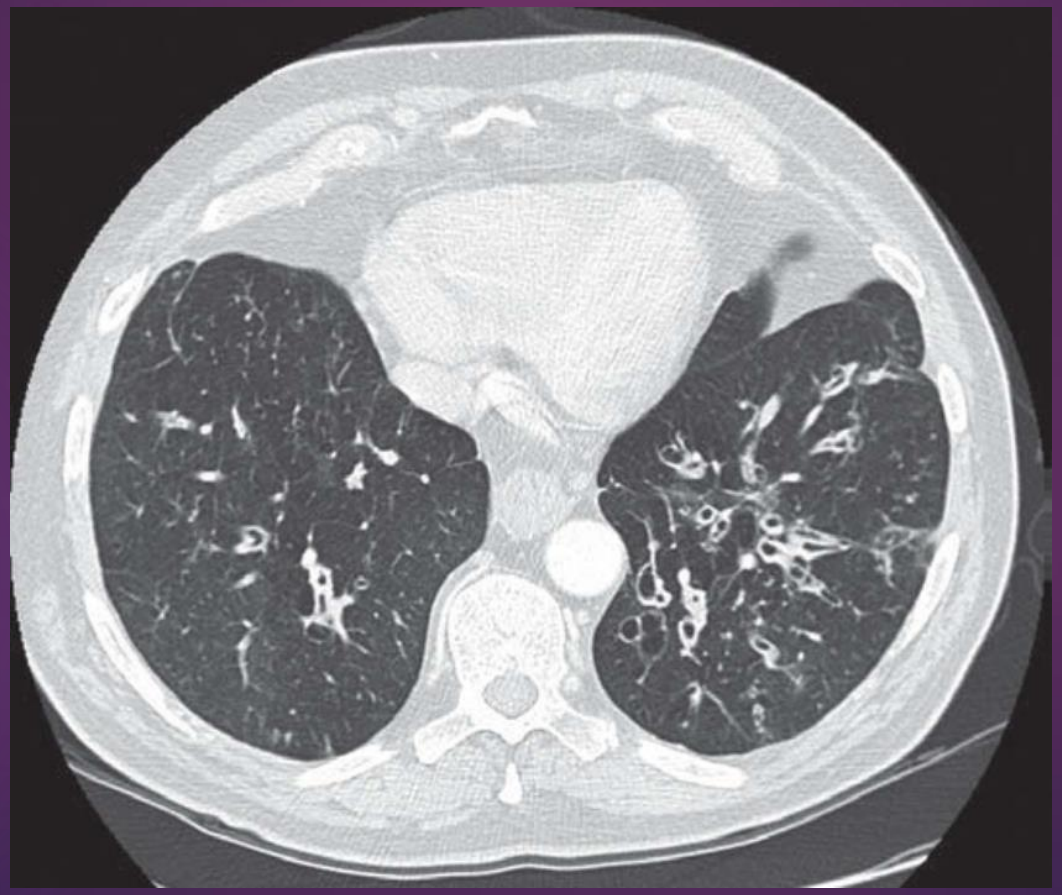
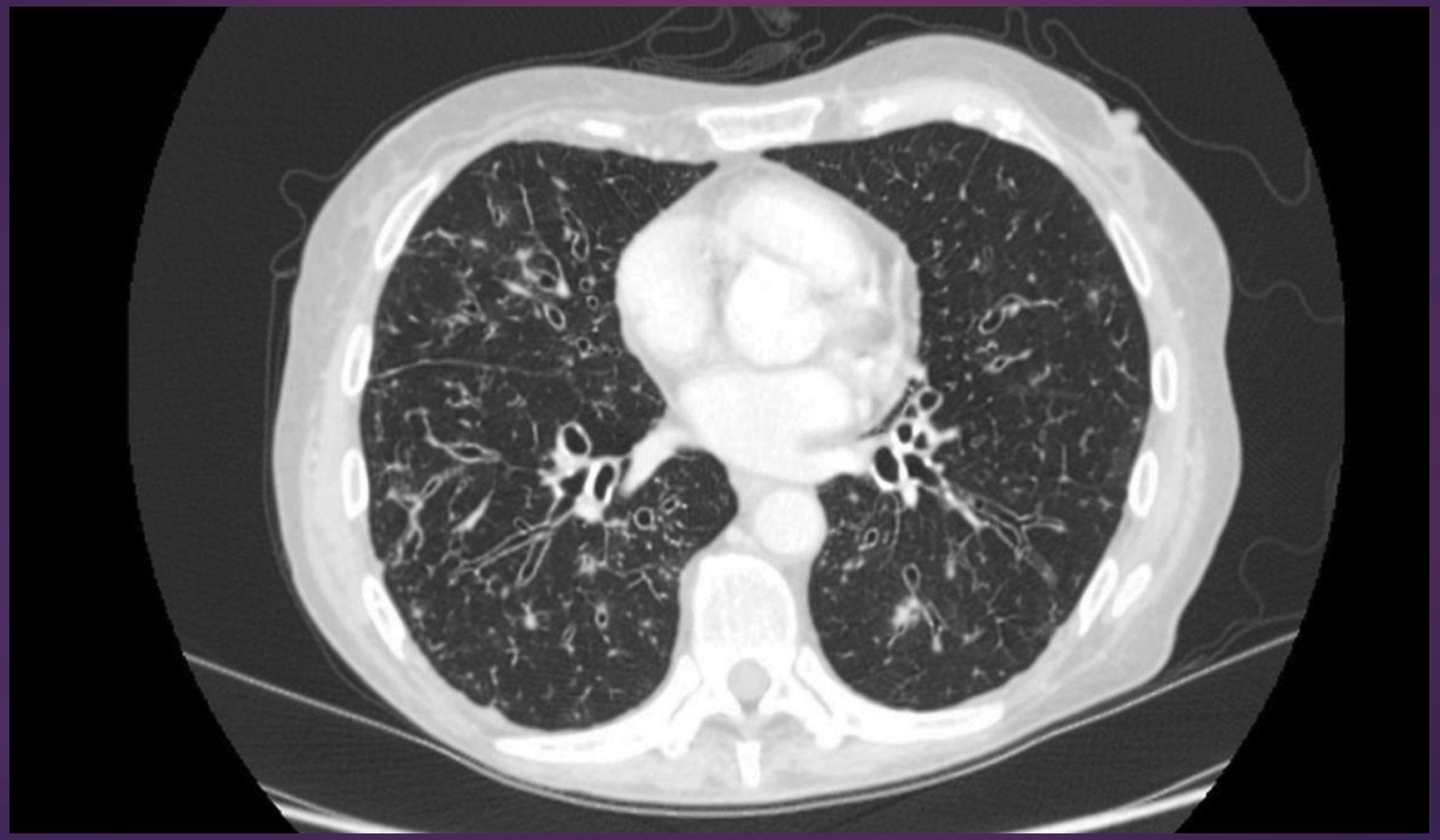
Bronchiectasis



Bronchiectasis



APPROACH TO THE PATIENT
Bronchiectasis

- ***clinical history***
- ***chest imaging***
- ***workup to determine the underlying etiology***

APPROACH TO THE PATIENT
Focal bronchiectasis

- Almost always requires bronchoscopy

- airway obstruction by an underlying mass or foreign body

APPROACH TO THE PATIENT
Diffuse bronchiectasis

- analysis for the major etiologies

- initial focus on excluding CF

- Pulmonary function testing

TREATMENT
Bronchiectasis

- control of active infection
- improvements in secretion clearance
- bronchial hygiene
  - decrease the microbial load within the airways
  - minimize the risk of repeated infections

ANTIBIOTIC TREATMENT

- Haemophilus influenzae
- P. aeruginosa
- acute exacerbations
  - 7–10 days and perhaps for as long as 14 days
- NTM infection can be difficult
  - two sputum samples positive on culture
  - one BAL fluid sample positive on culture;
  - a biopsy sample displaying histopathologic features of NTM infection
    - granuloma or a positive stain for acid-fast bacilli
  - one positive sputum culture; + pleural fluid sample positive on culture
- macrolide-sensitive MAC
  - macrolide combined with rifampin and ethambutol

BRONCHIAL HYGIENE

- hydration
- mucolytic administration
- bronchodilators
- hyperosmolar agents (e.g.,hypertonic saline),
- chest physiotherapy (postural drainage,mechanical chest percussion
- Pulmonary rehabilitation and a regular exercise program
  - improved exercise capacity and quality of life
- mucolytic dornase (DNase)
  - CF-related bronchiectasis

ANTI-INFLAMMATORY THERAPY
Glucocorticoids

- inhaled glucocorticoids
  - alleviated dyspnea
  - decreased need for inhaled β-agonists
  - reduced sputum production — ***no significant differences***
  - ***lung function***
  - ***bronchiectasis exacerbation rates***

oral/systemic Glucocorticoids

-  Risks of immunosuppression
- adrenal suppression

- **ABPA**
- **noninfectious bronchiectasis**
  - Rheumatoid arthritis
  - Sjögren's syndrome

- ABPA
  - oral antifungal agent itraconazole

REFRACTORY CASES

- Surgery
  - resection of a focal area of suppuration
- In advanced cases → lung transplantation

COMPLICATIONS

- In more severe cases
- recurrent infections & repeated courses of antibiotics

can lead to

- microbial resistance to antibiotics
- combinations of antibiotics
- life-threatening hemoptysis
  - intubation
    - identification of the source of bleeding
    - protection of the nonbleeding lung
  - bronchial artery embolization and, in severe cases, surgery

PROGNOSIS

- Outcomes of bronchiectasis
  - Underlying etiology
  - comorbid conditions
  - frequency of exacerbations
  - specific pathogens involved
    - P. aeruginosa colonization
- clinical, radiographic, and microbial features
- Assessment of quality of life and disease severity
- FEV1 declining by 50–55 mL per year
  - 20–30 mL per year for healthy controls

PREVENTION

- **Reversal of an underlying immunodeficient state**
  - administration of gamma globulin for immunoglobulin-deficient
- **vaccination**
  - influenza and pneumococcal vaccines
- **Smoking cessation**

TREATMENT

- After resolution of an acute infection
- in patients with recurrences (≥3 episodes per year)

6–12 months

- suppressive antibiotics
  - frequency of exacerbations
  - mucus production
  - decline in lung function

antibiotic resistance ?

macrolide-resistant NTM
rule out NTM infection
rule out a prolonged QT interval

TREATMENT

- (1) administration of an oral antibiotic
  - ciprofloxacin daily for 1–2 weeks per month;
- (2) use of a rotating schedule of oral antibiotics
- (3) administration of a macrolide antibiotic daily or 3 times/ week
  - anti-inflammatory effects
  - reduction of gram-negative bacillary biofilms
- (4) inhalation of aerosolized antibiotics (tobramycin inhalation solution)
  - 30 days on, 30 days off
- (5) intermittent administration of IV antibiotics (“clean-outs”)
  - more severe bronchiectasis and/or resistant pathogens

122 Lung Abscess

Rebecca M. Baron, Miriam Baron Barshak

Lung abscess

- necrosis and cavitation of the lung following microbial infection.

- single
- multiple

- single dominant cavity >2 cm in diameter

ETIOLOGY

- The low prevalence
- Significant morbidity and mortality

- primary (~80% of cases)
- Secondary

ETIOLOGY
Primary lung abscesses

- Aspiration
  - anaerobic bacteria
  - absence of an underlying pulmonary or systemic condition

ETIOLOGY
Secondary lung abscesses

- setting of an underlying condition
  - Post obstructive process (bronchial foreign body or tumor)
  - systemic process (e.g., HIV infection or another)

Lung abscesses

- Acute (<4–6 weeks in duration)
- Chronic (~40% of cases)

EPIDEMIOLOGY

- middle-aged men > middle-aged women
- risk factor for primary lung abscesses is **aspiration**
  - altered mental status
  - Alcoholism
  - drug overdose,
  - Seizures
  - bulbar dysfunction
  - prior cerebrovascular or cardiovascular events
  - neuromuscular disease
  - esophageal dysmotility or esophageal lesions (strictures or tumors)
  - gastric distention and/or gastroesophageal reflux
  - ***gingivitis and periodontal disease***

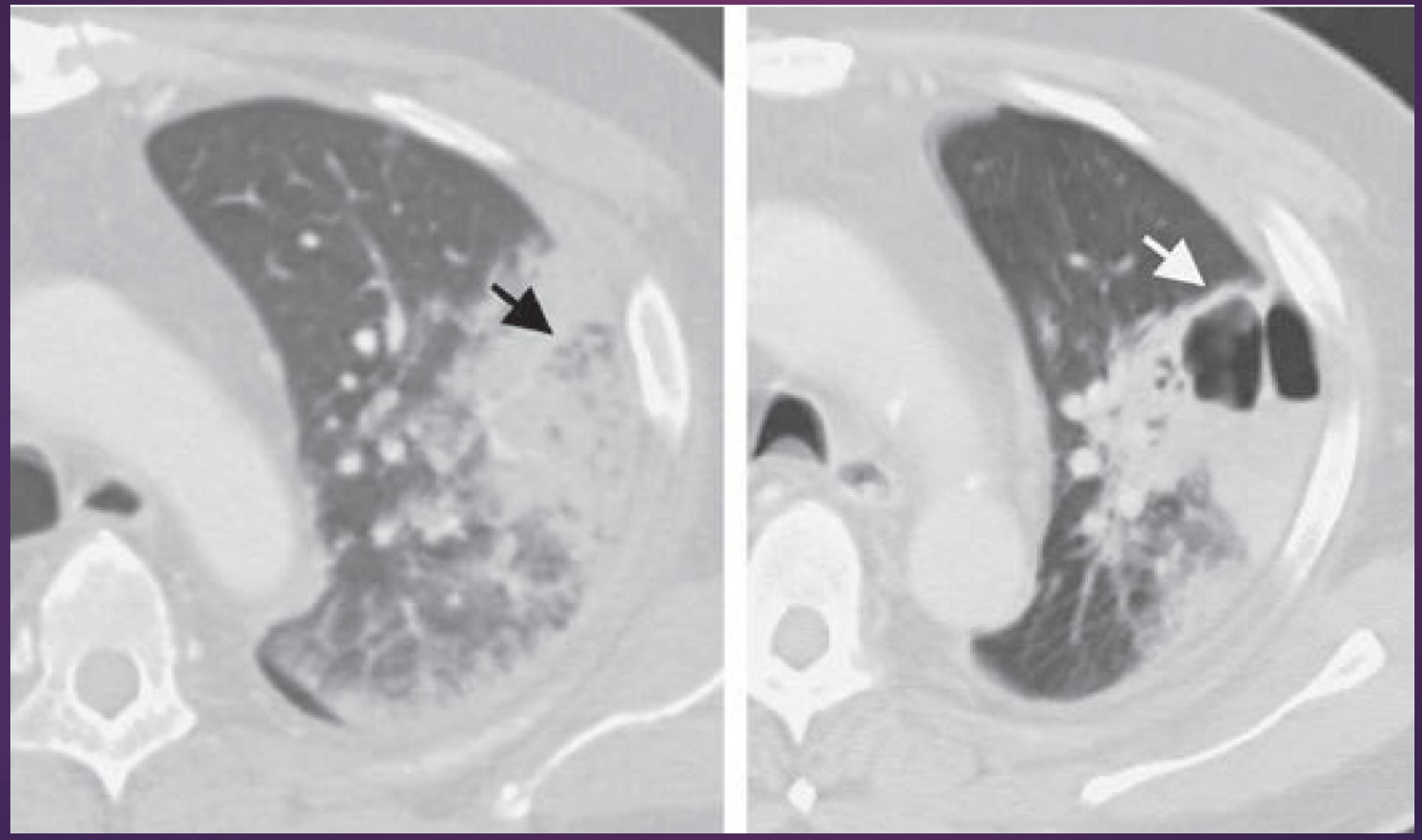
Aspiration

- Pneumonitis develops initially (tissue damage caused by gastric acid
- over a period of 7–14 days, the anaerobic bacteria
  - parenchymal necrosis and cavitation
  - depends on host–pathogen interaction
- Anaerobes
- Polymicrobial infections
  - synergistically to cause more significant tissue destruction

Primary lung abscess (usually with risk factors for aspiration)

- Anaerobes
  - Peptostreptococcus spp.,
  - Prevotella spp.,
  - Bacteroides spp.,
  - Streptococcus milleri),
- microaerophilic streptococci

underlying lymphoma
severe Pseudomonas aeruginosa pneumonia



Secondary Lung Abscesses

- **predisposing factor**
  - bronchial obstruction from malignancy or a foreign body
  - prevents clearance of oropharyngeal secretions,
    - leading to abscess development
- **underlying systemic conditions**
  - immunosuppression after bone marrow or solid organ transplantation
  - impaired host defense mechanisms
    - opportunistic organisms
- **septic emboli**
  - tricuspid valve endocarditis (often involving Staphylococcus aureus)
  - Lemierre's syndrome
    - infection begins in the pharynx (Fusobacterium necrophorum)
    - spreads to the neck and the carotid sheath (which contains the jugular vein)
    - cause septic thrombophlebitis.

Secondary Lung Abscesses with underlying immunocompromise

- Staphylococcus aureus
- gram-negative rods
  - Pseudomonas aeruginosa
  - Enterobacteriaceae
- Nocardia spp.
- Aspergillus spp.
- Mucorales
- Cryptococcus spp.
- Legionella spp.
- Rhodococcus equi,
- Pneumocystis jirovecii

Secondary Lung Abscesses

- **Endemic infections**
  - Mycobacterium tuberculosis
    - Mycobacterium avium
    - Mycobacterium kansasii
  - Coccidioides spp
  - Histoplasma capsulatum
  - Blastomyces spp
  - parasites
    - Entamoeba histolytica
    - Paragonimus westermani
    - Strongyloides stercoralis

Miscellaneous conditions

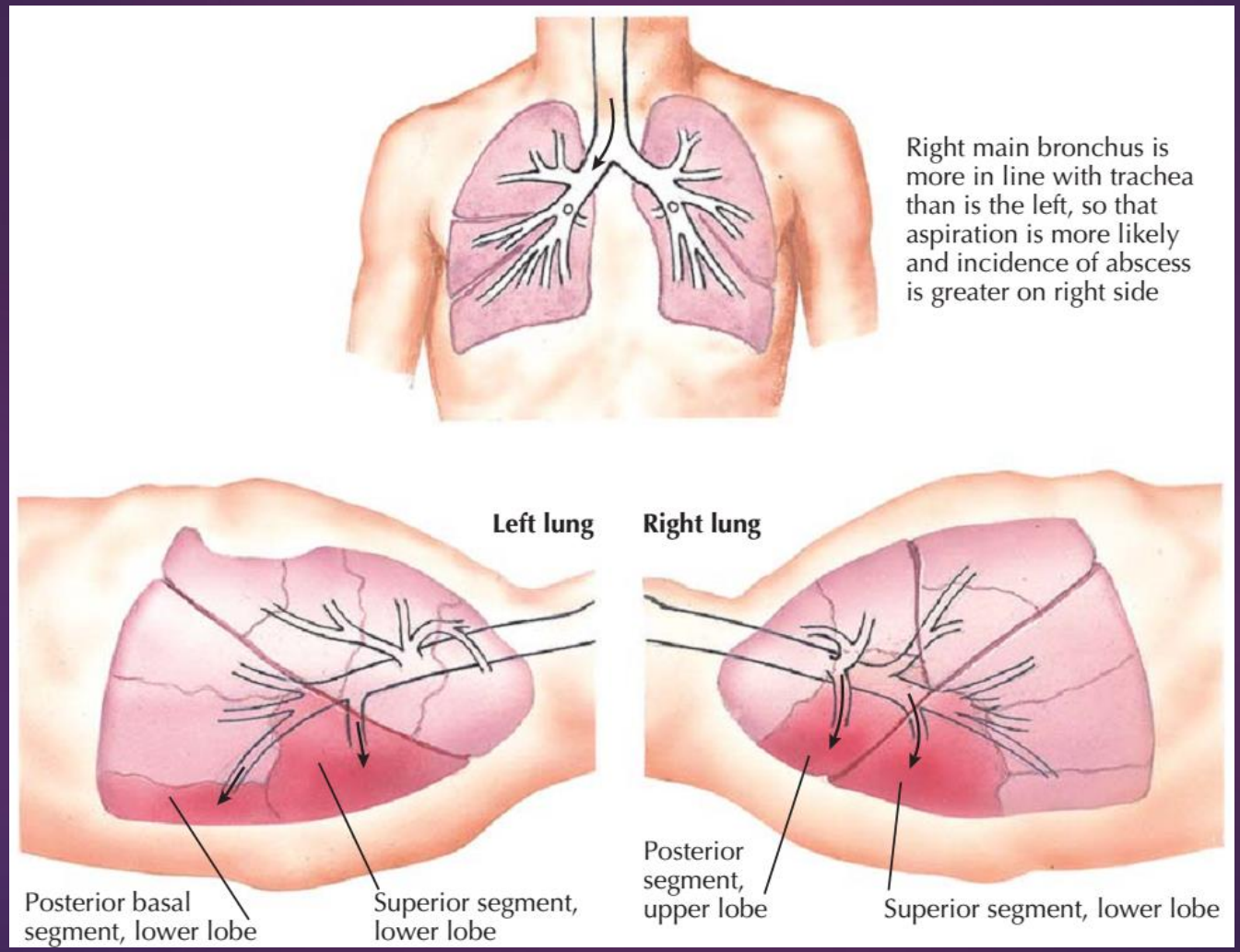
- Bacterial pathogen (often S. aureus) after influenza or another viral
- Actinomyces spp

PATHOLOGY AND MICROBIOLOGY
Primary Lung Abscesses

- The dependent segments
- Posterior upper lobes
- superior lower lobes
- right lung is affected more commonly

- often polymicrobial,
  - anaerobic organisms
  - Microaerophilic streptococci

A.P


Right main bronchus is more in line with trachea than is the left, so that aspiration is more likely and incidence of abscess is greater on right side
Left lung
Right lung
Posterior basal segment, lower lobe
Superior segment, lower lobe
Posterior segment, upper lobe
Superior segment, lower lobe

PATHOLOGY AND MICROBIOLOGY
Primary Lung Abscesses

- A putrid lung abscess
- foul-smelling breath, sputum, or empyema;

- anaerobic lung abscess

PATHOLOGY AND MICROBIOLOGY
Secondary Lung Abscesses

- location of secondary abscesses may vary with the underlying cause
  - Pseudomonas aeruginosa and other gram-negative rods
  - fungal infections among immunosuppressed patients

- immunocompromised hosts
  - unusual organisms

CLINICAL MANIFESTATIONS lung abscesses

- similar to those of pneumonia,
  - fevers, cough, sputum production, and chest pain
- a more chronic and indolent presentation anaerobic lung abscesses
  - night sweats, fatigue, and anemia
- putrid lung abscesses
- discolored phlegm
- foul-tasting or foul-smelling sputum
- Due to non-anaerobic organisms, such as S. aureus
  - More fulminant course high fevers and rapid progression

physical examination

- Fevers
- poor dentition, and/or gingival disease
- amphoric and/or cavernous breath sounds on lung auscultation.
- digital clubbing
- absence of a gag reflex

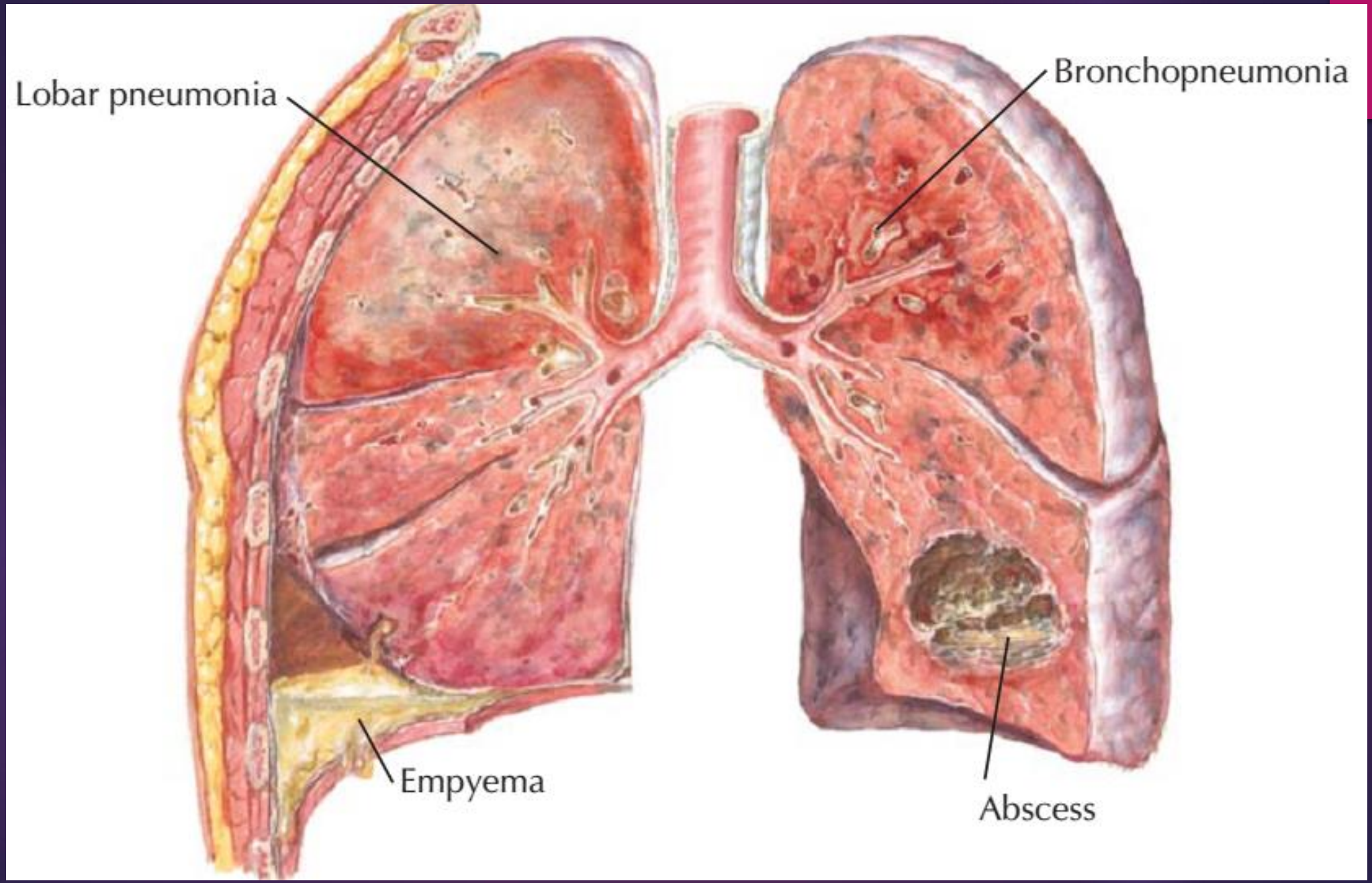
DIFFERENTIAL DIAGNOSIS

- broad & noninfectious processes that result in cavitary lung lesions
- lung infarction
- Malignancy
- Sequestration
- cryptogenic organizing pneumonia
- Sarcoidosis
- vasculitides and other autoimmune diseases
  - granulomatosis with polyangiitis
- lung cysts or bullae containing fluid
- septic emboli (e.g., from tricuspid valve endocarditis).
- pulmonary manifestations of diseases other than the chest
  - inflammatory bowel disease
  - pyoderma gangrenosum

DIAGNOSIS

- chest imaging
- thick-walled cavity with an air-fluid level,

- CT may also yield additional information
- Malignancy
- distinguish a peripheral lung abscess from a *pleural infection*
  - *require urgent drainage*


Lobar pneumonia
Bronchopneumonia
Empyema
Abscess

DIAGNOSIS

- more invasive diagnostics (such as transtracheal aspiration)
- Empirical therapy that includes drugs targeting anaerobic organisms

- polymicrobial, and culture results may not reflect

- putrid-smelling sputum
  - anaerobic infection

DIAGNOSIS

- secondary lung abscess
- empirical therapy fails

- sputum and blood cultures
  - viruses and fungi
- Bronchoscopy with BAL or protected brush specimen collection
  - spillage of abscess contents into the other
- CT-guided percutaneous needle aspiration
  - pneumothorax
  - bronchopleural fistula

- secondary abscesses, especially in immunocompromised hosts,

TREATMENT
Lung Abscess

- 1940s and 1950s
- 3–4 weeks to as long as 14 weeks
- (1) clindamycin
- (2) IV-administered β-lactam/β-lactamase combination
- 3 - moxifloxacin (400 mg/d PO)
- Metronidazole is not effective as a single agent:
  - not the microaerophilic streptococci

TREATMENT
secondary Lung Abscess

- **identified pathogen, and a prolonged course**
- if the primary lung abscess fails to improve

additional studies to rule out

- underlying predisposing cause for a secondary lung abscess

- 10–20% of patients may not respond at all
  - continued fevers and progression of the abscess cavity on imaging
  - surgical resection
  - percutaneous drainage of the abscess
- An abscess >6–8 cm in diameter is less likely to respond to antibiotic
- complications of percutaneous drainage
  - Bacterial contamination of the pleural space
  - Pneumothorax
  - Hemothorax

COMPLICATIONS

- Larger cavity size
  - Persistent cystic changes (pneumatoceles)
  - Bronchiectasis

- recurrence of abscesses
- Empyema
- life-threatening hemoptysis
- massive aspiration of lung abscess contents

PROGNOSIS AND PREVENTION

- mortality rates for primary abscesses have been as low as 2%,
  - secondary abscesses 75%
- poor prognostic
  - Age of >60
  - presence of aerobic bacteria
  - sepsis at presentation
  - symptom duration of >8 weeks
  - abscess size of >6 cm.

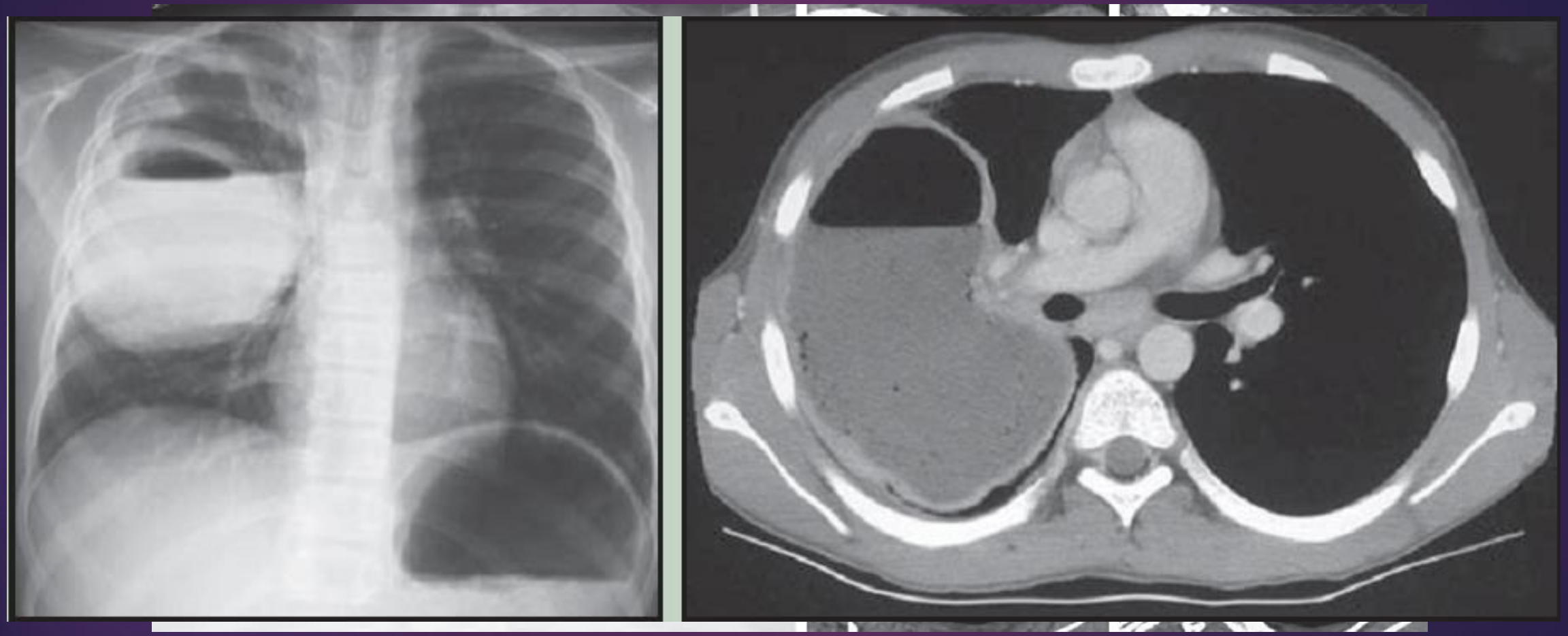
PROGNOSIS AND PREVENTION

- underlying risk factors ➡ prevention of lung abscesses
- Airway protection
- oral hygiene
- minimized sedation
- elevation of the head of the bed
- Prophylaxis against certain pathogens in at-risk patients
  - bone marrow or solid organ transplants
  - HIV infection

APPROACH TO THE PATIENT
Lung Abscess

- For patients with a lung abscess
  - low likelihood of malignancy (smokers <45 years old)
  - with risk factors for aspiration
- empirical treatment and then to pursue further evaluation if therapy does not elicit a response
- ***risk factors for malignancy***
- ***underlying conditions (especially immunocompromised hosts)***
- ***atypical presentation,***
- earlier diagnostics should be considered
- bronchoscopy with biopsy or CT-guided needle aspiration.
  - consistent with possible bronchial obstruction.
- endemic for tuberculosis or patients with other risk factors for tuberculosis
  - induced sputum samples

Lung abscess



CA-MRSA, oral anaerobes, endemic fungi, M. tuberculosis, atypical mycobacteria